

MY APPROACH to the use of NOACs for stroke prevention in patients with atrial fibrillation

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DOI:

[10.1016/j.tcm.2014.05.012](https://doi.org/10.1016/j.tcm.2014.05.012)

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Peer reviewed version

Citation for published version (Harvard):

Lip, GYH 2014, 'MY APPROACH to the use of NOACs for stroke prevention in patients with atrial fibrillation', *Trends in Cardiovascular Medicine*, vol. 24, no. 6, pp. 265-266. <https://doi.org/10.1016/j.tcm.2014.05.012>

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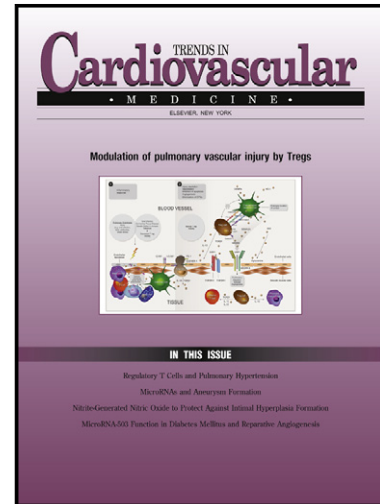
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PII: S1050-1738(14)00046-2
DOI: <http://dx.doi.org/10.1016/j.tcm.2014.05.012>
Reference: TCM6000

To appear in: *trends in cardiovascular medicine*

Cite this article as: Gregory Y H Lip MD, FRCP, DFM, FACC, FESC, MY APPROACH to the use of NOACs for stroke prevention in Patients with atrial fibrillation, *trends in cardiovascular medicine*, <http://dx.doi.org/10.1016/j.tcm.2014.05.012>

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MY APPROACH to the Use of NOACs for Stroke Prevention in Patients With Atrial Fibrillation



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Commentary

Stroke prevention is central to the management of atrial fibrillation (AF). The approach is to initially identify patients at a low risk for stroke (CHA₂DS₂-VASc score of 0 [men] or 1 [women]), who do not need any antithrombotic therapy. Subsequent to this step, patients with \geq additional stroke risk factors (thus, CHA₂DS₂-VASc score of ≥ 2 , as well as men with a score of 1) can be offered effective stroke prevention.

Effective stroke prevention essentially means oral anticoagulation (OAC), whether given as a well-controlled, adjusted-dose vitamin K antagonist (VKA; eg, warfarin), or one of the non-VKA oral anticoagulants (NOACs; previously referred to as new, or novel, OACs¹). If VKAs are used, the challenge is to maintain patients within an international normalized ratio (INR) of 2.0 to 3.0. Guidelines recommend good-quality anticoagulation control, as reflected by an average individual time in therapeutic range (TTR) of $\geq 70\%$.^{2,3} The TTR correlates with the efficacy and safety of VKAs whereby a high TTR is associated with low risks for thromboembolism and bleeding, but a low TTR (ie, $<60\%$) is associated with a high risk for thromboembolism or hemorrhage.^{3,4}

For newly diagnosed anticoagulation-naïve patients with AF, use of the SAME-TT₂R₂ score can help identify those patients (SAME-TT₂R₂ score of 0–2) who are likely to do well with a VKA (with TTR $\geq 70\%$). On the other hand, patients with a SAME-TT₂R₂ score >2 are less likely to do well with a VKA and require more intense review and INR monitoring; therefore, they may be better off with an NOAC.⁵ We do not practice a "warfarin stress test" whereby patients are initiated with a VKA for 3 to 6 months, and only if the TTR is suboptimal would they be permitted to switch to an NOAC. Such an approach leads to the initial period whereby patients in the inception cohort would have suboptimal TTRs and would lead to a substantial risk for fatal and devastating strokes.⁶

In an established patient who is already receiving anticoagulation with a VKA, clinical follow-up includes a review of the INRs recorded in the anticoagulation record booklet. If the TTR is suboptimal (ie, $<60\%$ despite efforts to improve anticoagulation control by our clinic), the patient would be considered for switching from a VKA to an NOAC. The VKA is stopped, the INR is

allowed to decrease to approximately 2.0, and the NOAC is started.^{7,8} Because all of the NOACs have a fast onset of action, no bridging is necessary.

With the availability of a number of NOACs, prescribers are now spoiled regarding choice, and we can fit the drug to a particular patient profile (and vice versa). For example, in a patient with a high risk for bleeding (HAS-BLED score ≥ 3), the NOACs that have a safer bleeding profile (eg, dabigatran 110 mg twice daily or apixaban) can be used. When renal function is impaired (eg, creatinine clearance ≈ 30 mL/L), it would be prudent to use an NOAC that is not so dependent on renal excretion, such as apixaban and rivaroxaban. Dabigatran should be avoided, given the high renal excretion of this drug. For maximal potency in reducing ischemic stroke (eg, in a patient with recurrent strokes despite reasonable TTR), dabigatran 150 mg twice daily can be considered.

My patients starting with any OAC are given a counseling or education session, focused on compliance and precautions about use of anticoagulants. Part of the counseling process is drug-specific (eg, advice not to take dabigatran on an empty stomach to avoid any dyspepsia). Also, rivaroxaban needs to be taken with food. Follow-up at intervals requires assessment of any changes in the clinical situation (eg, bleeding problems) and monitoring of renal function.

Abbreviations

SAMe-TT ₂ R ₂	Sex female; Age <60 years; Medical history (>2 comorbidities); Treatment (interacting medications; eg, amiodarone); Tobacco use (doubled); Race (doubled)
CHADS ₂	Congestive heart failure; Hypertension; Age ≥ 75 years; Diabetes; previous Stroke
CHA ₂ DS ₂ -VAsC	Congestive heart failure, Hypertension, Age ≥ 75 years, Diabetes, previous Stroke, Vascular disease, Age 65-74 years, Sex category (female)
HAS-BLED	Hypertension; Abnormal renal/liver function; Stroke; Bleeding history or predisposition; Labile international normalized ratio; Elderly (>65 years); Drugs/alcohol concomitantly
VKA	Vitamin K antagonist
TTR	Time in therapeutic range
OAC	Oral anticoagulation

NOAC

Non-VKA oral anticoagulant

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